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(1)

AD-A196 377

REPORT DOCUMENTATION PAGE		READ INSTRUCTIONS BEFORE COMPLETING FORM
1. REPORT NUMBER AFIT/CI/NR 88-3	2. GOVT ACCESSION NO.	3. RECIPIENT'S CATALOG NUMBER
4. TITLE (and Subtitle) EFFECT OF PHYSICAL FITNESS ON RESPONSE TO ORTHOSTASIS IN HEALTHY YOUNG WOMEN		5. TYPE OF REPORT & PERIOD COVERED MS THESIS
		6. PERFORMING ORG. REPORT NUMBER
7. AUTHOR(s) CAROLYN K. GOOCH		8. CONTRACT OR GRANT NUMBER(s)
PERFORMING ORGANIZATION NAME AND ADDRESS AFIT STUDENT AT: UNIVERSITY OF WASHINGTON		10. PROGRAM ELEMENT, PROJECT, TASK AREA & WORK UNIT NUMBERS
CONTROLLING OFFICE NAME AND ADDRESS		12. REPORT DATE 1988
		13. NUMBER OF PAGES 67
MONITORING AGENCY NAME & ADDRESS (if different from Controlling Office) AFIT/NR Wright-Patterson AFB OH 45433-6583		15. SECURITY CLASS. (of this report) UNCLASSIFIED
		15a. DECLASSIFICATION/DOWNGRADING SCHEDULE

DISTRIBUTION STATEMENT (of this Report)

DISTRIBUTED UNLIMITED: APPROVED FOR PUBLIC RELEASE

DTIC ELECTE AUG 0 3 1988 S D H

17. DISTRIBUTION STATEMENT (of the abstract entered in Block 20, if different from Report)

SAME AS REPORT

18. SUPPLEMENTARY NOTES

Approved for Public Release: IAW AFR 190-1
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Dean for Research and Professional Development
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19. KEY WORDS (Continue on reverse side if necessary and identify by block number)

20. ABSTRACT (Continue on reverse side if necessary and identify by block number)

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THE EFFECT OF PHYSICAL FITNESS
ON RESPONSE
TO ORTHOSTASIS IN HEALTHY YOUNG WOMEN

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Master of Nursing, 1988; 75 pages

The purpose of this study was to examine the relationship between fitness level and cardiovascular response to orthostatic stress in healthy women between the ages of 20 and 35. Subjects were divided into three groups on the basis of VO_2max: High-Fit (n = 4; mean VO_2max = 57.8 ml/kg/min); Med-Fit (n =8; mean VO_2max = 46.4 ml/kg/min); and Low-Fit (n = 4; mean VO_2max = 34.7 ml/kg/min). Subjects were exposed to three trials of 70° head up tilt each followed by a ten minute supine rest period. Heart rate and blood pressure were recorded at 1, 3 and 5 minute intervals during head up tilt and at 5 and 10 minute intervals during the supine rest period. The mean heart rate and blood pressure changes from supine to one minute upright were calculated for each group. The change in heart rate per change in systolic blood pressure ($\Delta HR/\Delta SBP$) from supine to 70° upright was used as an index of baroreflex responsiveness. There was a strong inverse correlation between fitness level and heart rate both resting and at 1 minute upright (r=.83;r=.84 $p < .001$). An analysis of baroreflex indicies between groups showed no fitness related differences in heart rate and blood pressure response to orthostasis.

The Effect of Physical Fitness

on Response to Orthostasis

in Healthy Young Women

by

Carolyn K. Gooch

A thesis submitted in partial fulfillment
of the requirements for the degree of

Master of Nursing

University of Washington

1988

Approved by Marie J. Cowan PhD
(Chairperson of the Supervisory Committee)

Program Authorized
to Offer Degree Nursing

Date May 26, 1988

Master's Thesis

Signature ____________

Date May 26, 1988

University of Washington

Abstract

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Chairperson of Supervisory Committee: Marie Cowan, Ph.D.

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ACKNOWLEDGMENTS

The author wishes to express sincere appreciation to Dr. Marie Cowan and Dr. William Ryan for their guidance and assistance in the preparation of this manuscript. In addition, special thanks to Professor Janet Marvin for professional critique of the prepared manuscript and to the Physiological Nursing Department for assistance with facilities and equipment.

CHAPTER 1

Problem Statement

Orthostasis, the act of assuming an upright posture, triggers a complex series of physiological mechanisms in the human body. Gravitational forces are responsible for the translocation of 500 to 800ml of blood primarily from the intrathoracic compartment to the vessels of the lower extremities (Gauer & Thron, 1965; Blomqvist & Stone, 1983). Downward displacement of blood results in a reduced cardiac filling pressure which is translated via the Starling mechanism into a diminished cardiac output and narrowed pulse pressure. Reduced distending pressures in the heart and subsequent narrowing of pulse pressure trigger a compensatory reflex designed to maintain arterial blood pressure and protect cerebral perfusion.

Baroreceptors in the heart, aorta, great veins, lungs and carotid arteries respond to decreased pressure by sending fewer signals centrally causing a reduction of parasympathetic inhibition and an augmentation of sympathetic input to the cardiovascular system. The normal physiological response to orthostasis, mediated by the autonomic nervous system, is an accelerated heart rate, an increase in peripheral vascular resistance, an elevation of circulating catecholamines and secretion of water retaining hormones (Blomqvist & Stone, 1983).

Passive standing, tilt-table manipulation, lower body negative pressure (LBNP) and centrifugation are research methods which have been developed to evaluate cardiovascular adjustment to upright posture. Hemodynamic parameters are measured before and after imposition of orthostatic stress to determine the physiological reaction to position change. Orthostatic measurement of vital signs is also useful clinically as a diagnostic procedure to evaluate certain circulatory abnormalities. A variety of factors can impinge on reflex compensatory cardiovascular mechanisms and result in orthostatic intolerance manifested primarily as postural hypotension. Hemorrhage, varicosities of the legs, high environmental temperatures, pharmacological vasodilation, heavy exercise, cardiac abnormalities, hypoxia, emotional stress, dehydration, and prolonged bed rest are some of the conditions which can predispose an individual to orthostatic hypotension by altering effective circulating volume (Schatz, 1984; Brevetti, Chiarello & Campenella, 1983; Ziegler, 1980). A second general category of orthostatic hypotension is caused by any interruption of the baroreceptor reflex and may include neuropathy, lesions in the neuronal pathway, aging, pharmacological agents that act on the autonomic nervous system, trauma, surgery and in rare cases excess circulating hormones (Vargas & Lee, 1982; Ibrahim, 1975;

Ibrahim, Tarazi & Dustan, 1975; Donald & Shepard, 1979).

Several researchers have attempted to determine constitutional factors responsible for altering the efficacy of orthostatic response. Orthostatic tolerance has been found to be independent of gender (Shvartz & Meyerstein, 1970) and weight (Klein, Bruner, Jovy, Vogt & Wegmann, 1969). An inverse relationship exists between orthostasis and age > 60 years (Robbins & Rubenstein, 1984: Smith et al., 1987) as well as height (Howard & Leathart, 1951). The literature is divided with regard to the relationship between physical fitness, measured by maximal oxygen uptake (VO_2max), and response to orthostatic stress. Several researchers have demonstrated what they believe to be a "hypotonia" or an attenuated autonomic reflex in endurance trained athletes which is exhibited in a diminished tachycardic response, an inefficient maintenance of systolic blood pressure, a smaller elevation of peripheral vascular resistance and a tendency to pool greater quantities of blood in the lower extremities (Stegemann, Busert & Brock, 1974; Klein, Wegmann & Kuklinski, 1977; Raven, Rohm-Young & Blomqvist, 1984; Smith & Raven, 1986).

Other comparisons of athletes to non-athletes find no significant difference in orthostatic response (Shvartz & Meyerstein, 1972; Convertino, Sather,

Goldwater & Alford, 1986; Frey, Mathes & Hoffler, 1987; Hudson, Smith & Raven, 1987). Convertino, Montgomery & Greenleaf (1984) found no alteration in orthostatic tolerance in a group of men whose VO_2max had been significantly increased after a short period of endurance training. Furthermore, they suggest that training actually enhanced the blood pressure response through a significant exercise induced increase in plasma volume. Hudson et al., (1987) found no difference in the overall hemodynamic response to -50 mmHG LBNP in trained and untrained women.

Nurses managing the care of either acutely or chronically ill patients need a clear understanding of the physiology involved in cardiovascular adjustment to postural changes, along with the ability to assess patient progress through increasing levels of activity during hospitalization. An important ancillary goal is the identification of those patients at high risk for developing complications as they resume normal activity after surgery or debilitating illness.

With the current emphasis on physical fitness and aerobic conditioning programs, the relationship between fitness level and postural changes gains relevance to nursing practice. Postural manipulation in-hospital falls primarily in the nursing domain as an independent intervention. Experimental evidence clarifying the

relationship between fitness and orthostasis may provide some basis for understanding one aspect of positioning as it relates to specific segment of the patient population.

Purpose

The purpose of this study is to describe and compare the blood pressure and heart rate response to orthostatic stress imposed by a 70° head up tilt in women between the ages of 20 and 35 designated as high-fit, medium-fit and low-fit on the basis of VO_2max.

CHAPTER II

Conceptual Framework

Regulation of arterial blood pressure and volume redistribution after postural changes is achieved through the concurrent operation of several mechanisms. The following is a discussion of blood volume redistribution following postural changes and principle hemodynamic regulatory mechanisms including: a) baroreceptor reflexes, b) humoral responses, and c) mechanical factors.

Blood Volume Distribution

Blood exists as a continuous column of fluid in a closed system. Volume is normally distributed with approximately 70% in systemic veins, 15% in the heart and lungs, 10% in the systemic arteries, and 5% in the capillaries (Rushmer,1970). In the supine position blood is exposed only to static pressure determined by volume and dynamic pressure generated by the pumping action of the heart. With a change from supine to upright posture the blood comes under the influence of gravity and is exposed to hydrostatic pressure expressed as pgh (where p = fluid density, g = acceleration due to gravity, and h is the height of the column of fluid) (Rowell, 1986). A pressure gradient is established in the vertical column of blood with arterial pressures ranging from 60mmHg in the cranium to 200mmHg in the

feet (Rowell, 1986). Upon standing, 500 to 600ml of blood flows downward into the legs while another 200 to 300ml is sequestered in the veins of the buttocks and pelvis (Rowell, 1986; Blomqvist & Stone, 1983). In an upright position approximately 70% of man's circulating blood volume is below the level of the heart (Rowell, 1986). Up to 78% of the blood shifting to dependent portions of the body is supplied by thoracic vessels (Gauer & Thron, 1965; Sjostrand, 1953). The immediate result of the downward translocation of blood is a fall in central venous pressure, resulting in a reduction in ventricular filling pressures, followed by a fall in cardiac output, stroke volume and arterial pulse pressure (Rowell,1986). The following is a discussion of the neurogenic, hormonal, and mechanical adjustments which function to restore cardiac filling pressures and support cardiac output in the upright position.

Hemodynamic Regulatory Mechanisms

The downward relocation of blood upon standing constitutes a loss to the central system analogous to a mild hemorrhage and triggers a sequence of compensatory adjustments aimed at preserving arterial blood pressure and tissue perfusion. Principle mechanisms which function to maintain blood pressure and volume distribution following postural changes include: a) autonomic reflexes mediated by arterial and

cardiopulmonary baroreceptors; b) humoral responses mediated by circulating hormones; and c) mechanical factors, primarily through muscular contraction and abdominothoracic pressure (Blomqvist & Stone, 1983).

Arterial Baroreceptors

Free nerve endings in the carotid sinus and the arch of the aorta respond to mechanical deformation (stretch or reduction of stretch) by increasing or decreasing the frequency of impulses sent to the cardiopulmonary center in the medulla (Blomqvist & Stone, 1983; Vander, Sherman & Luciano, 1985; Berne & Levy, 1986). Afferent impulses from the carotid baroreceptors ascend through the sinus and glossopharyngeal nerves to synapse in the nucleus tractus solitarius (NTS) of the medulla. Impulses from the aortic arch ascend via the vagal nerves. An increase in baroreceptor stretch caused by added pressure stimulates the firing frequency of afferent nerve fibers causing inhibition of sympathetic outflow to the cardiovascular system via efferent fibers to the sinoatrial node, ventricles, veins, and arterioles; parasympathetic outflow to the heart is augmented (Blomqvist & Stone, 1983; Berne & Levy, 1986; Rowell, 1986). Conversely, a decrease in pressure at the baroreceptors stimulates a pressor response.

Stroke volume is reduced by as much as 40% on standing (Marshall & Shepard, 1968). The resulting narrowed pulse pressure causes a decreased impulse frequency to the medulla resulting in increased sympathetic outflow to the heart and peripheral vasculature. Parasympathetic inhibition of the heart is diminished. The immediate result is increase in cardiac contractility and rate along with peripheral vasoconstriction.

Cardiopulmonary Baroreceptors

Low pressure receptors located in the atria, ventricles and pulmonary veins respond to changes in intracardiac, central venous or pulmonary vascular pressure. Impulses ascend to the medullary control center via the vagus, reflexively increasing or decreasing peripheral vascular tone (Blomqvist & Stone, 1983). The heart rate response is negligible. In addition to causing peripheral vasoconstriction, a drop in pressure at the cardiopulmonary receptors initiates important humoral responses in defense of plasma volume. These are discussed in the following section.

Humoral response.

Certain hormones are released or inhibited systemically as part of the baroreceptor reflex. The most important of these in the context of postural hemodynamics are vasopressin (ADH), renin, and

aldosterone. ADH is released from the posterior lobe of the pituitary in response to a reduction in pressure at atrial baroreceptors (Rowell, 1986). Although it has potent vasoconstrictor capabilities in large concentrations, the most important function of ADH in humans is the effect it has on water absorption in the distal tubules of the kidney. A reduction in cardiopulmonary baroreceptor output following downward redistribution of circulating volume is associated with an increase in renal sympathetic drive which triggers the release of renin.

Renin facilitates the conversion of angiotension I to the vasoactive hormone angiotensin II which plays a dual role in defense of pressure through direct vasoconstriction and stimulation of aldosterone release from the adrenal medulla. Aldosterone in turn facilitates the absorption of sodium and water from the distal tubules of the kidney in defense of plasma volume. Increased distention of the cardiopulmonary and arterial baroreceptors associated with a rise in pressure suppresses the release of both renin and ADH (Epstein, 1978).

Mechanical Factors

In addition to baroreceptor and humoral responses, certain mechanical factors operate to maintain blood pressure during postural changes. Venous anatomy,

muscular contraction and respiratory mechanics all contribute to the regulation of arterial blood pressure (Rushmer, 1970). The following is a discussion of the contribution of tissue pressure and abdominothoracic pressure to the maintenance of arterial blood pressure and volume distribution.

Tissue pressure; the "muscular pump". Muscular contraction during upright posture plays a large part in maintaining central venous filling pressures and consequently systemic arterial pressure. Contraction of the leg muscles compresses the deep veins resulting in an expulsion of blood headward and a reduction of peripheral venous pressure. With the relaxation of the muscle, venous valves help to retard the return of the blood to dependent extremities although with prolonged standing these valves will open and blood will once again form a continuous column (Rowell, 1986). Simple contraction and relaxation of the calf muscles while standing can return stroke volume to nearly supine levels (Wang, Marshall & Shepard, 1960).

Abdominothoracic pressure; the "respiratory pump". Intrathoracic pressure becomes increasingly negative with inspiration while intra-abdominal pressure rises as the diaphragm descends. The combined pressures enhance the filling of intrathoracic vessels and the emptying of abdominal vessels during inspiration

(Rushmer, 1970; Rowell, 1986). During expiration the process is reversed and repetition of the process results in a "pumping" action.

Normal Cardiovascular Response to Orthostasis

The immediate physiological response to acute posture change is a fall in central venous pressure and activation of the sympathetic nervous system mediated by baroreceptors. The resulting accelerated heart rate and increased peripheral vascular resistance maintain mean arterial pressure at close to supine levels.

Passive head up tilt and active standing from the supine position result in similar hemodynamic adjustments (Wolthuis, Bergman & Nicogossian, 1974). Wang et al. (1960) measured cardiac output, heart rate and stroke volume in seven healthy young men and found a 41% fall in stroke index with standing. Light exercise consisting of contraction of the calf muscles was sufficient to raise the stroke index to near supine levels. Ward, Danziger, Bonica, Allen, and Tolas (1966) studied 20 healthy adults while supine, standing and sitting and found that standing caused a 45% decrease in stroke volume, a 27% decrease in cardiac output, along with increased heart rate, mean arterial pressure and total peripheral resistance. Glezer and Moskalenko (1972) measured cardiovascular hemodynamics in recumbent and upright positions and found a moderate decrease in

systolic pressure, an increase in diastolic pressure, with no change in mean arterial pressure. In addition to pressure changes, they recordeo an increase in stroke index and heart rate and total peripheral vascular resistance increased. Circulating blood volume decreased and hematocrit increased. Lance and Spodick (1977) made non-invasive measurements of postural changes in cardiovascular volumes and dynamics. Comparing supine to standing, they found upright posture increased heart rate, isovolumic contraction time, pre-ejection period and pre-ejection period/left-ventricular ejection time along with decreases in ejection time and ejection time index.

Head Up Tilt

Active standing, passive tilt, lower body negative pressure (LBNP) and centrifugation have been used to impose orthostatic stress for purposes of research. These methods have also been employed clinically to diagnose autonomic dysfunction and volume deficiencies. Measurement of the circulatory adjustment to gravity is influenced by the method used to place the subject in an upright position because even slight movement of leg muscles increases venous return to the heart (Wang et al., 1960).

Qualitatively similar results to standing and passive head up tilt are well documented in the

literature. Schvartz and Meyerstein (1970) tilted 36 healthy young men and women to an angle of 70° on a table with a padded motorcycle seat for crotch support for a total of 20 minutes or until syncope and noted elevations in heart rate and diastolic blood pressure, a slight lowering of systolic blood pressure and a narrowing of pulse pressure. Matalon and Farhi (1979) utilized a tilt table with a foot rest to tilt subjects from 0 to 90° noting a 40% decrease in cardiac output, a 27% increase in heart rate, and a 50% reduction in stroke volume. Using non-invasive measurement techniques, Spodik, Meyer, and Pierre (1971) demonstrated a 19% increase in heart rate and significant reduction in cardiac volumes occurring in the first minute of tilt.

Orthostatic Tolerance

Researchers have determined orthostatic tolerance to be independent of gender (Shvartz & Meyerstein, 1970); and weight (Klein, Bruner, Vogt & Wegmann, 1969); inversely related to height (Shvartz, 1968); and differentially related to the time of day (Harma & Lansimies, 1985). The concept of orthostatic intolerance is generally understood in terms of an inadequate autonomic response to upright posture clinically manifested as orthostatic hypotension (Schatz, 1984). Since crew members on Skylab 4 with different levels of

aerobic fitness exhibited varying responses to orthostatic stress, there has been a suggestion that high fitness levels may predispose to orthostatic intolerance (Frey, 1987). The following section is a discussion of conflicting evidence concerning the relation of physical fitness to orthostatic tolerance.

Physical Fitness and Orthostatic Tolerance

In the current literature physical fitness is most often defined as aerobic power and described in terms maximal oxygen uptake (VO2max) during exercise. Maximal aerobic power is defined as "the highest oxygen uptake the individual can attain during physical work while breathing air at sea level" (Åstrand & Rodahl, 1977). Recent research has suggested that endurance training resulting in high aerobic fitness (VO_2max) may be detrimental to the maintenance of blood pressure with postural changes. Stegmann, Bursert and Brock (1974) compared the blood pressure control system of 25 endurance trained athletes to that of 25 non-athletes by changing transmural pressure in the carotid artery using a sealed pressure chamber (modified "iron lung" originally used to artificially ventilate polio patients). The pressure in the chamber was altered from -60 to +60 mmHg while heart rate and blood pressure were recorded. Athletes were found to have *significantly* smaller heart rate and blood pressure changes in

response to externally applied changes in carotid artery transmural pressure. The authors concluded that this attenuated circulatory response would be disadvantageous for blood pressure regulation during orthostasis especially after any additional stressor such as bedrest, blood loss or weightlessness. Raven, Rohm-Young and Blomqvist (1984) suggest that the diminished cardiovascular response during orthostasis seen in endurance athletes is a reflection of "blunted" autonomic control mechanisms. After exposing 8 fit (VO_2max=70.2 ± 2.6 ml/kg/min) and 6 average fit (VO_2max=41.3 ± 2.9 ml/kg/min) to -50 Torr of lower body negative pressure, fit subjects showed a significantly lower tachycardic response; 7% compared to 11% for average fit. Fit subjects also exhibited a lower peripheral vascular resistance along with a greater drop in systolic blood pressure and a significantly smaller increase in HR for a given fall in systolic BP ($\Delta HR/\Delta SBP$). Raven et al. (1984) concluded that endurance training altered the physiological reflex response to the downward shift of fluid following orthostasis.

A similar effect was demonstrated by Smith and Raven (1986) in the comparison of cardiovascular responses of 8 sedentary, 8 endurance trained and 8 weight trained subjects to -50 Torr of lower body

negative pressure. Endurance trained subjects were the least effective in maintaining blood pressure during the hypotensive challenge. The calculated index of barorecptor sensitivity ($\Delta HR/\Delta SBP$) was significantly lower for endurance trained subjects. In a study relating bed rest deconditioning effects to aerobic fitness, Goldwater et al. (1980) measured plasma renin activity in 12 healthy men after 10 days of bed rest. High athletic conditioning (on a continuum of VO_2max from 43.4 to 23.9 ml/kg/min.) was associated with lower plasma renin activity and hypotension after bed rest.

Results documented by other researchers contradict the theory that physical fitness has a negative effect on orthostatic tolerance. Klein et al. (1969) compared responses of 12 untrained (VO_2max= 43.9 ml/kg/min) to 12 highly trained (VO_2max= 64.9 ml/kg/min) to a 90° head up tilt of 20 minutes and found no difference in orthostatic intolerance measured by the number of fainting episodes. More recently Convertino, Montgomery, and Greenleaf (1984) compared orthostatic response to head-up tilt in the same individuals before and after 8 days of endurance training. Following training VO_2max increased by 8.3%, resting heart rate decreased by 8.1% and plasma volume increased by 12.2%. In response to tilting, systolic blood pressure, diastolic blood pressure and mean arterial pressure were

sustained even though heart rate response was lower than pre-training levels. In addition to maintenance of blood pressure, six of eight subjects were able to withstand an increased tilt duration after training. On the basis of these findings Convertino et al. (1984) suggest that orthostatic tolerance is actually augmented by endurance training and that reflex response may be more accurately related to the rate of plasma volume loss and pooling of blood in the extremities. In a subsequent study, Convertino, Sather, Goldwater & Alford (1986) noted the point at which syncope occurred during exposure to LBNP. They could establish no correlation between syncope and physical fitness.

Summary

Cardiovascular adjustment to gravitational stress depends on rapid activation of complex homeostatic mechanisms. Compensation for downward fluid shift is accomplished through increases in heart rate and vascular tone mediated by the autonomic nervous system. A variety of pathological conditions can interfere with compensatory mechanisms resulting in orthostatic instability. Inefficient adjustment to orthostatic stress is seen primarily as a failure to maintain adequate blood pressure in the upright position. Postural hypotension is usually indicative of some functional or neurogenic problem. In addition to

pathology, some constitutional characteristics seem to be related to differential orthostatic tolerance. Some researchers have documented a diminished orthostatic response with aging, while no negative relationship has been determined with gender or weight. Evaluation of the relationship between physical fitness, defined by maximal oxygen uptake, and orthostatic tolerance has yielded conflicting results. The purpose of this study is to examine and compare the heart rate and blood pressure responses of highly fit, medium fit and low fit women when exposed to orthostatic stress.

CHAPTER III

Methods

Design

This study used a pretest-postest design to compare the effects of physical fitness level on physiological response to orthostasis in three groups of women. After determination of individual aerobic fitness level utilizing indirect calorimetry during a treadmill test (see Appendix A for test protocol), subjects were divided into three groups according to maximal oxygen consumption (VO_2max). Orthostatic challenge was simulated with a 70° head up tilt. Dependent variables measured were heart rate and blood pressure recorded at five and ten minutes prior to head up tilt and at one, three and five minutes during upright position.

Sample

The sample consisted of 16 healthy women between the ages of 20 and 35 selected from a convenient sample of students at the University of Washington and the general public from the surrounding metropolitan area of Seattle.

A notice soliciting participants was posted on central bulletin boards throughout the University of Washington campus. Respondents were interviewed over the phone using a short questionnaire to determine eligibility for the study. Items on the questionnaire covered age, gender, weight, height, current and past

health status including any use of medications, smoker or non-smoker status, and subject's evaluation of her fitness level (see Appendix B). Specific entrance criteria were formulated to minimize the influence of extraneous variables on study results and to safeguard the health of the subjects.

Inclusion Criteria

1) Healthy: Defined as a negative history for physical or psychiatric illness and no current evidence of disease or functional disability.

2) Female.

3) Between the ages of 20 and 35.

Exclusion Criteria

1) Smoker: Smoking is known to alter oxygenation and blood pressure (Colby, 1977).

2) Currently taking medications or drugs of any kind (with the exception of birth control pills). Pharmacolgic agents can alter the autonomic response to postural changes (Schatz, 1984).

3) Pregnant: Exercise testing to maximum capacity during pregnancy is not recommended (American College of Sports Medicine, 1987).

4) Greater than 20% above recommended weight for height as defined by the Metropolitan Life

Insurance Table of Weights for women (see Appendix C). Obesity is often associated with elevated blood pressure (Colby, 1977) and could interfer with accurate measurement if the spygmomanometer cuff fits improperly (Frank et al., 1973).

5) Taller than 5" 10" without shoes: Increased height has been associated with orthostatic intolerance (Klein et al., 1968).

Determination of Aerobic Fitness Level

Screening to determine VO_2max was conducted in the pulmonary function lab at University Hospital in Seattle, Washington with the primary researcher and an expert nurse advisor present. Current guidelines of the American College of Sports Medicine do not require the presence of a physician during maximal exercise testing in healthy adults $\leq$35 years old (see Appendix D).

Subjects were tested using a standard Bruce treadmill protocol (Bruce, Kusumi & Hosmer, 1973). VO_2max was measured by analysis of expired air with a Sensormedics Model 4400 computerized metabolic measurement cart previously calibrated with reference gases. Criteria for determination of maximal effort during the exercise test included the following:

a) Plateau of VO_2 values at the end of test.

b) Attainment of >85% of age predicted maximal

heart rate computed as 220 minus age as estimate (American College of Sports Medicine Guidelines, 1987).

c) Subjective rating of perceived maximal effort.

On the basis of VO_2max scores, subjects were divided into three groups: High-fit (n=4; VO_2max=57.8ml/kg/min); Med-fit (n=8; Avg.VO_2max=46.4ml/kg/min); and Lo-fit (n=4; Avg/VO_2max=34.7ml/kg/min).

Protection of Human Subjects

Volunteers for this study were thoroughly briefed concerning possible risks involved in the experimental protocol. Each subject was screened as to health status to eliminate any individual with a history of health problems or currently undergoing medical treatment. Since the subjects were young healthy women the risks were minimal but included the possibility of hypotension during implementation of the head up tilt and fatigue and muscle soreness following exercise testing. At the onset of any signs that suggested impending syncope and/or significant hypotension during the tilt procedure the experiment would have been terminated and the subject returned to a supine position. No significant hypotensive episodes occured. Data gathered were designated by code number only and kept under lock and key by the primary researcher.

Measurements

Head Up Tilt

Tilt table manipulation to elicit orthostatic response was conducted at the physiological laboratory of the University of Washington School of Nursing within 14 days of the VO_2max determination. Head up tilting was accomplished using a manually operated tilt table consisting of a padded platform with a foot support. Loose restraining straps across the abdomen, chest and thighs provided safety for the subject and served to minimize the use of postural muscles during upright posture (Smith, Hudson & Raven, 1987). The table was tilted to a 70° upright angle within two seconds to stimulate a strong autonomic response to position change (Sundkvist & Lilja, 1983). Each subject underwent three same day tilt trials, separated by ten minute rest periods to allow for return to baseline hemodynamic equilibration (Lance & Spodick, 1977).

Heart Rate Analysis

Heart rate was measured elecrocardiographically with a Hewlett Packard single lead monitor, model #1500 B, using a Lead II electrode placement. Recordings were taken during the last 12 seconds of minutes 5 and 10 during the supine rest period prior to each tilt and continously during each five minute tilt. Heart rate was calculated from the ECG strip using the R-R interval and

recorded at 30 second, 1, 3 and 5 minute points.

Blood Pressure Measurement

Blood pressure was measured indirectly at 5 and 10 minutes supine and 1, 3 and 5 minutes upright using the Critakon, Dynamap ™ Vital Signs Monitor Model # 1846. The right arm was used for blood pressure determination on every subject and the phlebostatic axis was marked prior to tilting to ensure the arm was supported at heart level for accurate blood pressure measurement (Frank et al.,1973).

Reproducibility

Because physiological response to orthostatic is a dynamic phenomenon subjects were tilted for a total of three trials to enhance reliability of measurements (Woods & Cantanzaro, 1988, p. 264). Heart rate and blood pressure scores all three tilts were averaged for each subject.

The difference between supine heart rate (HR) and HR at 1 minute upright divided by the difference between supine systolic blood pressure (SBP) and SBP at 1 minute upright (ΔHR/ΔSBP) was used as an index of barorecptor response (BR) (Smith & Raven, 1986). The BR index was computed for each tilt. The median BR index for three trials was selected as the strongest measure of centrality for each individual (Woods & Cantanzaro, 1988, p. 389).

Protocol

After determination of eligibility each subject received a thorough description of the experimental protocol including the nature of possible risks involved. Written consent was obtained prior to implementation of the study (see Appendix E). The consent form was endorsed by the University of Washington Human Subjects Committee.

On the day selected for determination of aerobic fitness level, subjects were instructed to report to the pulmonary functions lab at the University Hospital in a two to three hour post-absorptive state having abstained from the use of alcohol or any other drug (with the exception of birth control pills) for at least 72 hours prior to testing. Subjects were further instructed not to exercise vigorously for at least 24 hours prior to aerobic fitness determination (see Appendix F).

On a day not more than 14 days following determination of aerobic fitness level, subjects were instructed to report to the physiology laboratory at the University of Washington School of Nursing in comfortable loose clothing, once again in a post-absorptive state, having abstained from drugs of any kind for at least 72 hours and exercise for at least 24 hours. The subject was asked to assume a supine position on the tilt table while instrumentation was completed and phlebostatic axis

was marked. After placement of the ECG leads and attachment of the automated spygmomanometer, a ten minute rest period was provided to allow for a return to normal resting heart rate and blood pressure levels. Blood pressure and heart rate were recorded twice during the rest interval to establish supine baseline levels.

After the ten minute supine rest period, the subject was tilted to a 70° head up position. Tilting was accomplished in no more than two seconds (Sundkvist & Lilja, 1983). Head up tilt was maintained for five minutes. Heart rate was measured electrocardiographically throughout the tilt and blood pressure measurements were recorded at 1, 3 and 5 minutes (see Appendix G for data gathering tool). After five minutes in the 70° head up tilt position, the subject was returned to a supine position and allowed to rest for ten minutes to provide for equilibration of heart rate and blood pressure. Heart rate and blood pressure were recorded twice during the second supine rest period at 5 and 10 minutes. The five minute head up tilt followed by a ten minute rest period was repeated two more times for a total of three head up tilt trials and four ten minute rest periods. Following the final ten minute rest period, recording instruments were removed and the subject was allowed to assume an upright posture progressing slowly through sitting, dangling and standing phases.

On standing, the subject was observed closely for any signs of hypotension or inability to adjust to the upright position. All data were collected by the primary researcher to eliminate interrater inconsistencies.

Data Analysis

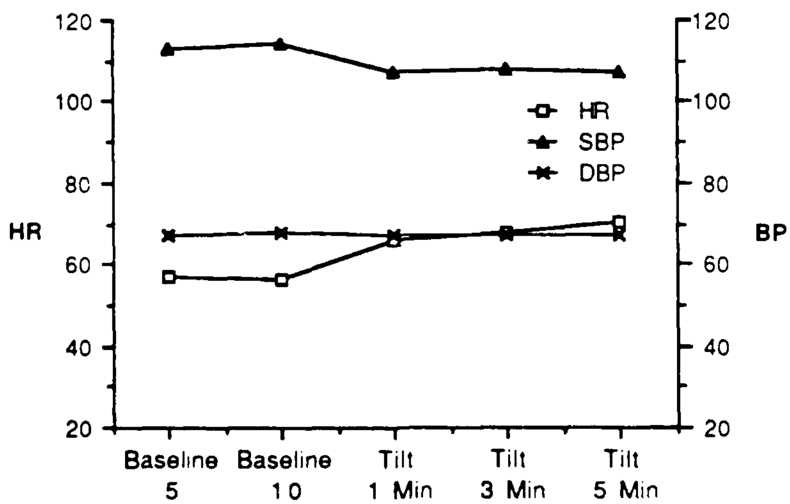
The study utilized a pretest-postest design and yielded descriptive data at the interval level after manipulation of the independent variable. A t-test for independent groups and a oneway ANOVA was used to compare differences in BR indicies between groups. Differences between HR and BP changes (measured in beats/minute and mmHg respectively) at 10 minute supine and one, three and five minutes tilt were examined using a non-parametric Kruskal-Wallis test. A Pearson correlation coefficient was used to test the relationship between VO_2max, HR and BP recorded at specified time intervals during supine and upright positions.

CHAPTER IV

Results

Average Individual BP and HR Response Changes

When heart rate and blood pressure response to tilt was averaged across three tilt trials for each subject the HR change from baseline to upright was +13.8% at 30 seconds, +16.9% at one minute, +21.5% at three minutes and +25.6% at five minutes. Mean SBP decreased by -6.5% at one minute, -4.9% at three minutes and -6.1% at five minutes. Mean DBP decreased slightly by -1.4% at one minute, -1.1% at three minutes and -1.4% at five minutes. Mean change in MAP was a decrease of -3.8% at one minute, -3.2% at three minutes and -3.8% at five minutes (see Table 1 page 30). Mean BP and HR response for the total sample is shown in figure 1.



<u>Figure 1.</u> Total sample blood pressure and heart rate response to 70° head up tilt at 1, 3, and 5 minutes.

TABLE 1
Average Heart Rate and Blood Pressure Changes
from Supine to 70° Head Up Tilt
for Total Sample

Subject ID	ΔHR bpm 30sec	ΔHR bpm 1min	ΔHR bpm 3min	ΔHR bpm 5min	ΔSBP mmHg 1min	ΔSBP mmHg 3min	ΔSBP mmHg 5min	ΔDBP mmHg 1min	ΔDBP mmHg 3min	ΔDBP mmHg 5min	ΔMAP mmHg 1min	ΔMAP mmHg 3min	ΔMAP mmHg 5min	Light-Headed 1min
1.	2.5	5.1	4.0	5.7	9	7	5	0	3	3	3	4	4	X
2.	1.1	3.7	4.3	6.1	7	9	6	4	5	7	5	6	7	X
3.	1.9	4.3	10.9	14.9	6	3	8	0	1	0	2	2	3	
4.	14.9	18.8	27.1	29.9	10	2	11	↑3	↑11	↑7	1	↑7	↑1	X
5.	↓1.4*	↓3.0	2.9	9.4	8	10	13	3	6	5	5	7	8	
6.	7.7	7.7	11.5	14.4	7	8	10	4	6	0	5	7	3	
7.	14.0	13.0	20.3	20.4	11	8	14	4	6	8	6	7	10	
8.	13.6	15.1	17.8	21.4	1	↑1	↑1	↑9	↑8	↑8	↑6	↑6	↑6	
9.	2.0	2.9	8.1	9.7	9	5	4	↑2	↑5	↑4	2	↑2	↑1	X
10.	3.8	3.5	4.3	5.7	3	9	3	15	13	17	11	12	12	
11.	13.0	13.3	16.4	15.2	8	↑2	3	2	0	↑1	4	↑1	0	X
12.	11.4	17.2	20.3	21.7	0	2	2	↑7	↑6	↑6	↑5	↑3	↑3	
13.	5.1	6.4	5.3	7.6	3	5	5	1	3	1	2	4	2	
14.	14.3	12.5	8.9	12.8	18	10	12	5	5	4	9	7	7	X
15.	13.5	15.5	19.7	18.9	11	8	12	↑4	↑4	↑5	1	0	1	
16.	↓0.2	9.9	1.2	6.1	8	6	6	4	5	6	5	5	6	
Mean	+7.3	+9.1	+11.4	+13.7	-7.4	-5.6	-7.2	-1.2	-1.2	-1.3	-3.3	-2.6	-3.3	
	±1.46	±1.50	±2.87	±1.74	±1.03	±0.92	±1.09	±1.34	±1.54	±1.55	±.81	±.66	±.81	
%Δ**	±13.8	+16.9	+21.5	+25.6	-6.5	-4.9	-6.1	-1.4	-1.1	-1.4	-3.8	-3.2	-3.8	
	±2.73	±2.87	±3.85	±3.72	±0.91	±0.77	±0.90	±1.92	±2.39	±2.34	±1.25	±1.55	±1.43	

* Where arrows are indicated the value differs in direction from the rest in that category.
** Percent change from baseline.

HR and BP Change by Group

Average age for the sample was 30.9 years. Mean weight and height were 59.7kg and 64.5cm respectively. Vo_2max ranged from 31.6 ml/min/kg to 63.1ml/min/kg. Subjects were divided into three groups on the basis of VO_2max: High-Fit (n=4 mean=57.8 ml/kg/min); Med-Fit (n=8 mean=46.4 ml/kg/min); and Low-Fit (n=4 mean=34.7 ml/kg/min). Table 2 covers descriptive data for the sample.

Table 2
Subject Descriptive Data

Subj. ID	Age yr	Wgt kg	Hgt cm	VO_2max ml/min/kg
		High-Fit		
1	32	56.2	64.0	63.1
2	28	55.8	66.0	58.7
3	29	50.8	64.0	57.1
4	29	62.1	68.0	52.1
		Med-Fit		
5	35	51.8	63.5	50.3
6	32	64.0	65.2	48.7
7	28	76.9	70.0	47.2
8	35	59.0	67.0	46.3
9	35	51.7	61.2	46.2
10	25	67.6	67.7	44.9
11	27	59.2	64.0	44.4
12	32	59.4	62.0	43.4
		Low-Fit		
13	34	58.1	62.0	39.0
14	32	51.3	61.5	35.9
15	32	67.7	65.0	32.3
16	29	63.5	63.0	31.6

Mean changes in heart rate and blood pressure for High-Fit, Med-Fit and Low-Fit groups compared to the means for the total sample are summarized in Table 3 on page 33. In this study hemodynamic changes at one minute are considered to be the most reflective of reflex autonomic adjustment to postural change (Guyton, 1987). Although there is a trend toward somewhat greater BP and HR changes in Low-Fit compared to High-Fit those differences are not significant. Figures 2 and 3 on page 34 and 35 provide a graphic illustration of HR and BP response to tilt for each group.

There was an inverse correlation between VO_2max and HR level at both baseline and one minute tilt ($r=-.84$; $r=-.85$ $p>.001$) (see figures 4 and 5 on page 36). No such correlation was evident between VO_2max and SBP or DBP. Fitness level was not significantly related to the magnitude of HR or BP change at any time interval ($p<0.05$).

Baroreceptor response index

The baroreceptor response (BR) index was computed to facilitate comparison between groups using the equation:

$$\frac{HR_2 - HR_1}{SBP_1 - SBP_2}$$

The BR index was computed for all three tilt trials for each subject. The median BR index was selected as the prefered index of central tendency in this study

TABLE 3
Average Heart Rate and Blood Pressure Changes
from Supine to 70° head up Tilt
for High-Fit, Med-Fit and Low-Fit Groups

	ΔHR* bpm				ΔSBP** mmHg			ΔDBP mmHg		
GROUP	30 sec	1 min	3 min	5 min	1 min	3 min	5 min	1 min	3 min	5 min
TOTAL	↑7.3	↑9.1	↑11.4	↑13.7	↓7.4	↓5.7	↓7.2	↓1.2	↓1.3	↓1.4
%Δ***	+13.8	+16.9	+21.5	+25.6	-6.5	-4.9	-6.1	-1.4	-1.1	-1.4
HIGH-FIT	↑5.1	↑8.0	↑11.6	↑14.2	↓8.3	↓5.5	↓7.5	↓0.3	↑0.5	↓0.8
%Δ	+11.8	+18.1	+25.8	+31.2	-7.1	-4.7	-6.6	-0.8	+2.1	-0.1
MED-FIT	↑8.0	↑8.7	↑12.7	↑14.7	↓5.9	↓4.9	↓6.1	↓1.3	↓1.5	↓1.5
%Δ	+15.2	+16.2	+23.6	+27.6	-5.1	-4.2	-5.0	-1.5	-1.8	-1.7
LOW-FIT	↑8.2	↑11.1	↑8.8	↑11.4	↓9.8	↓7.5	↓9.0	↓2.0	↓2.3	↓1.5
%Δ	+12.9	+16.9	+13.1	+16.9	-8.7	-6.5	-7.6	-2.5	-3.1	-2.1

* Heart rate change from baseline in beats per minute.
** Blood pressure change from baseline in millimeters of mercury.
*** Percent change from baseline

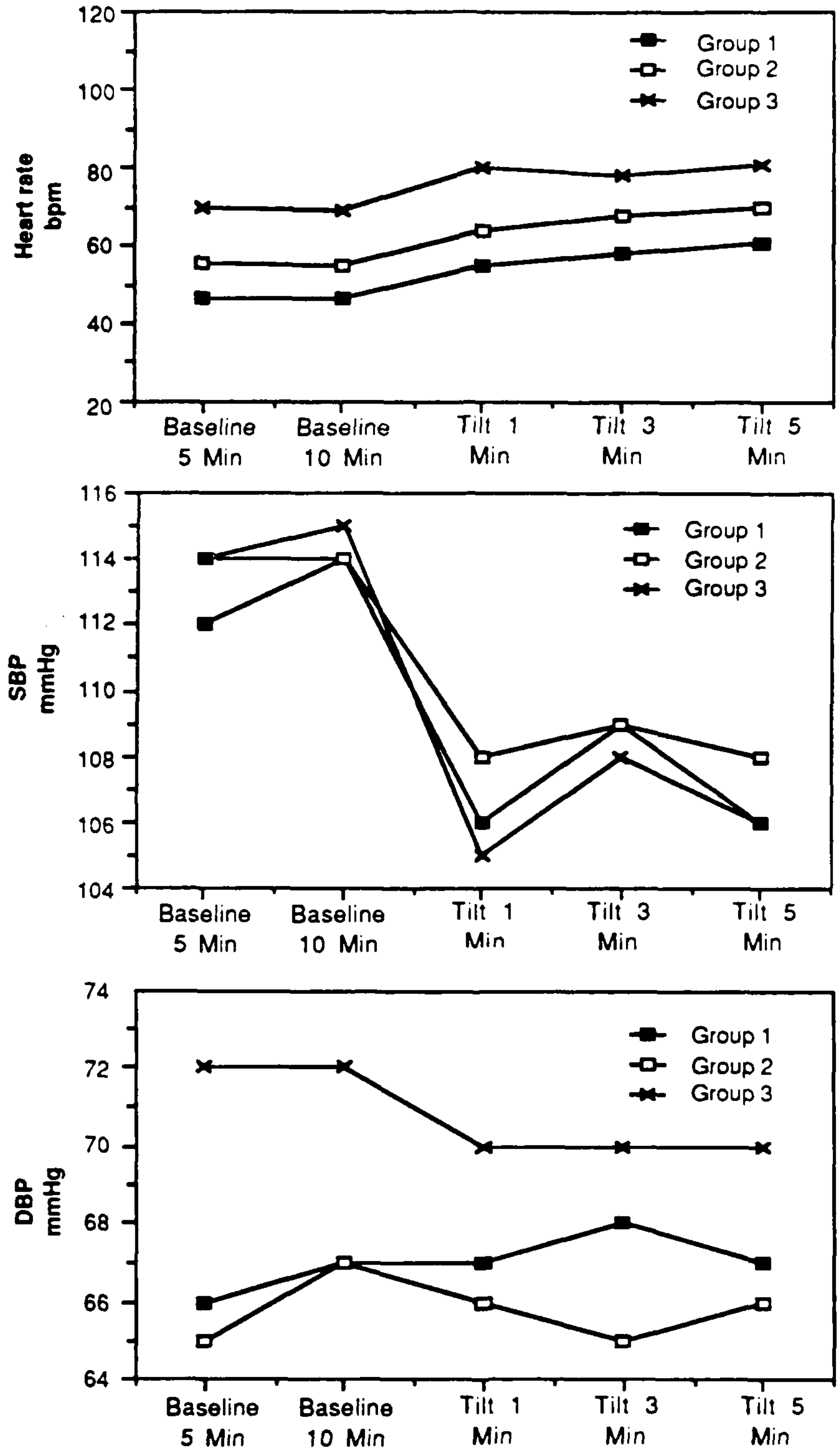


Figure 2. Dynamic blood pressure and heart rate response to tilt in High-Fit (1) Med-Fit (2) and Low-Fit (3) groups.

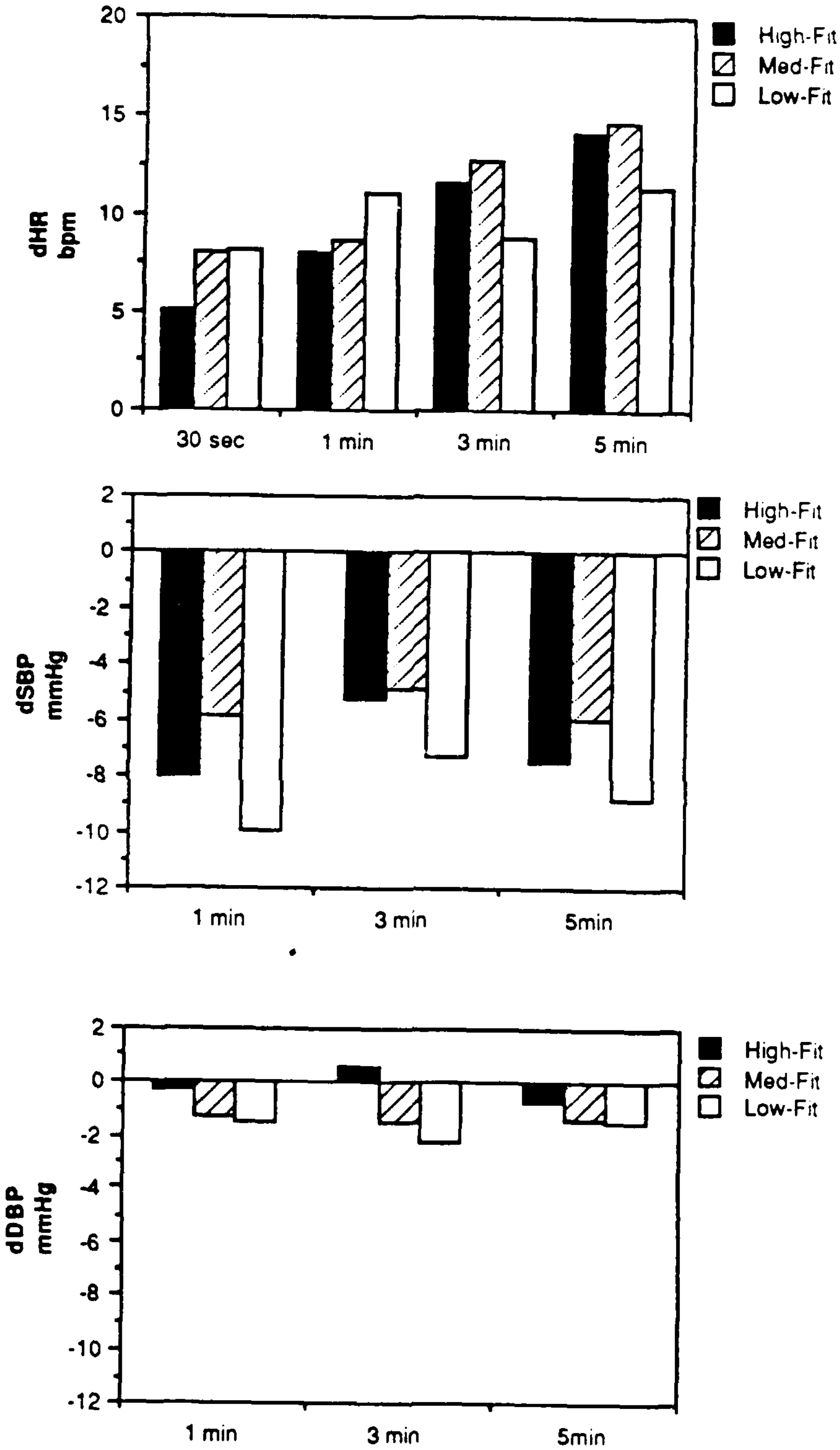
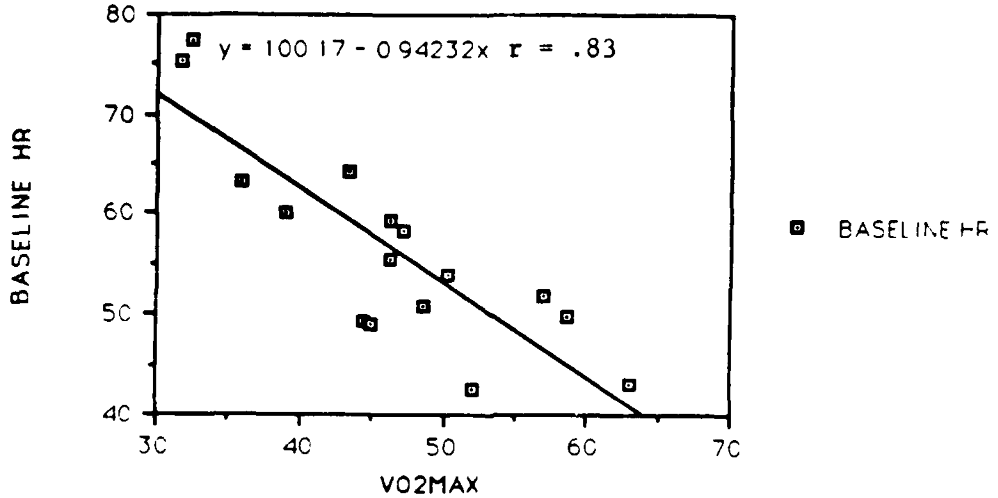
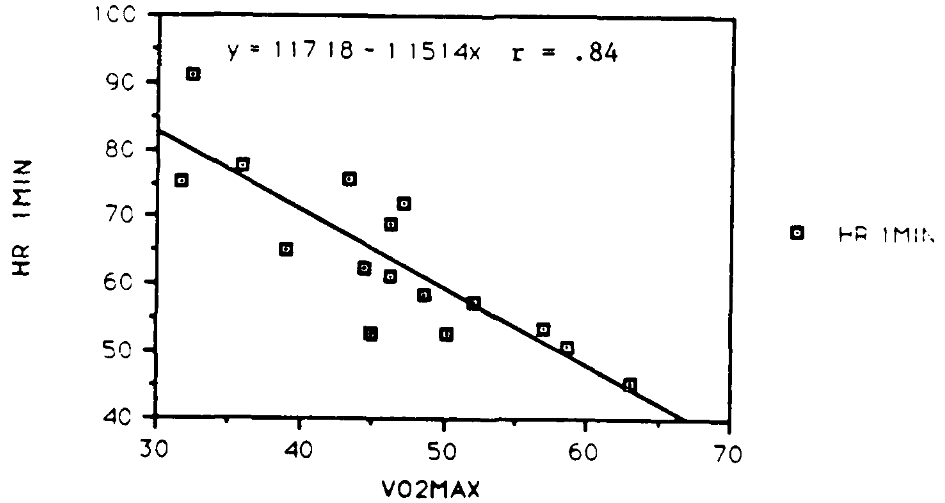


Figure 3. Group heart rate and blood pressure changes at 2 minute intervals during 70° head up tilt.



<u>Figure 4</u>. Baseline heart rate as a function of VO_2max



<u>Figure 5</u>. Heart rate at 1 minute 70° head up tilt as a function of VO_2max

because the distribution for several individuals was skewed (Polit & Hungler, 1983, pg. 477). Resulting values were: High-Fit=1.05 (±.22); Med-Fit=1.40 (±.97); and Low-Fit=1.25 (±.32). An analysis or variance between groups failed to reveal significant differences (see Appendix H).

BR indices of HighFit and Low-Fit subjects were compared in an effort to look more closely at differences beteen the two groups most divergent in fitness levels. Statistical comparison failed to demonstrate a significant difference between these two group indices (Appendix I).

Since the sample was small and therefore might not have been characterized by normality (Woods & Cantanzaro, 1988) a non-parametric Kruskal-Wallis statistical test was used to examine the difference in BR scores of all three groups. No significant difference between groups was demonstrated (Appendixs J and K).

General Hemodynamic Response to Tilt

The following is a description of the general dynamic response to tilt for all subjects. Each subject underwent three five minute 70° head up tilt trials. At one minute, out of a total of 48 tilt trials for 16 subjects, HR increased from supine to 70° upright by 0.9 to 27.4 beats per minute in 43 (86%) trials and

decreased from 0.1 to 11.5 beats per minute in five (10.4%) trials. The trials in which HR decelerated represented three subjects or 18.8% of the total sample (n=16). In two of these three cases deceleration was transient with HR rising to above resting levels by the fifth minute of tilt. In 44 (96.7%) trials SBP dropped from 1 to 24 mmHg below resting levels. In two (4.2%) trials SBP was greater than resting levels by 2 and 6 mmHg. DBP at one minute fell in 26 (54.2%) trials by 1 to 16 mmHg, was elevated in 17 (35.4%) trials from 1 to 10 mmHg and remained the same in five (10.4%) trials.

Greatest HR and SBP changes were exhibited in two (12.5%) subjects on the first tilt trial, six (37.5%) on the second tilt and eight (50%) in the third and final tilt. Six out of 16 subjects reported transient dizziness lasting approximately 10 to 20 seconds during the onset of the tilt prodecure. Four out of the six reported dizziness at the onset of the first tilt trial but did not experience dizziness in subsequent trials. Two subjects reported dizziness lasting from 10 to 20 seconds in all three tilts. These transient presyncopal episodes were not consistently associated with a particular pattern of blood pressure or heart rate response. Dizziness accompanied HR changes ranging from ↓0.1 to ↑27.4 beats per minute and SBP ranging from ↑1 to ↓24 mmHg.

CHAPTER V

Discussion

Assumption of upright posture and exposure of body fluid to gravitational forces is known to cause a pooling of blood in the lower extremities and a subsequent reduction in venous return to the heart (Gauer & Thron, 1965; Rushmer, 1970; Blomqvist & Stone, 1983). Diminished cardiac filling pressures and a fall in cardiac output initiate a complex set of homeostatic mechanisms mediated by arterial and cardiopulmonary baroreceptors (Ward et al., 1966; Gauer, Henry & Behn, 1970,). The net result is an increase in cardiac rate and contractility along with a rise in systemic vascular resistance (Shvartz, et al., 1982; Rowell, 1986).

Recent studies have suggested a connection between endurance training and a diminished autonomic response to orthostasis. Mechanisms variously cited as causing orthostatic intolerance in athletes are: excess fluid accumulation in the lower extremities (Klein et al., 1969); attenuated baroreflex sensitivity (Stegemann et al., 1974); a decreased ability to vasoconstrict (Raven et al., 1984); an alteration in the balance of the autonomic nervous system toward parasympathetic dominance (Smith & Raven, 1986); and lower plasma renin activity (Goldwater et al., 1980).

This study failed to demonstrate a significant fitness related difference in cardiovascular response to orthostasis in females. These findings agree with those of Frey, Mathes & Hoffler (1987) who found no difference in aerobic capacity between female subjects who fainted with exposure to -50 mmHg LBNP and those who did not. Hudson, Smith & Raven (1987) in a similar experiment evaluated 16 women during progressive LBNP found no difference in the baroreflex response ($\Delta HR/\Delta SBP$) between trained (VO_2max 56.8 ml/min/kg) and untrained (VO_2max 39.4 ml/min/kg) women.

Aerobic fitness in the current study was not correlated with any resting or upright parameter except baseline HR both supine and at one minute upright tilt. This finding coincides with previous research documenting significantly lower resting and tilt HR levels in athletes (Klein et al., 1969; Convertino et al., 1984; Härmä & Länsimies, 1985)

It has been suggested that the cardiovascular response occurring in the first minute of tilt reflects the most significant autonomic adjustment to postural changes (Guyton, 1981). Heart rate and blood pressure were selected in this study as the most representative of these changes and the most readily measured. Although no significant differences were noted in HR and BP changes at one minute between High-Fit, Med-Fit and

Low-Fit groups, some interesting trends emerged. If an accelerated HR is necessary to compensate for a reduction in cardiac output with standing then the slower initial acceleration seen in the High-Fit group might be explained by the relative hypervolemia and greater stroke volume known to be associated with high levels of aerobic fitness (Hill, Hill, Grisham & Zauner, 1987). Another interesting trend was the sustained rise in HR noted in the High-Fit compared to Low-Fit group (see Figure 4). A similar tendency was noted by Borst et al., (1982) who documented a continuing HR rise in trained compared to no such sustained rise in untrained subjects. The mechanism underlying this difference is not yet understood.

The slightly higher DBP in Low-Fit subjects may reflect greater resting sympathetic tone than High-Fit subjects in whom greater parasympathetic tone is characteristic (Winder, Hagberg & Hickson, 1978). Calf circumference was not measured in the current study. Therefore no statement can be made with regard to possible venous pooling effects.

Over-all cardiovascular response of women to upright posture in this study was qualitatively similar to that reported elsewhere in literature. The average maximum HR increase of +25.1%, and decreases of 6.1% in SBP and -0.5% in DBP seen in this study fell within

reported parameters which document HR increases from +13 to +37% (Ward et al., 1966; Chobanian et al., 1974); SBP changes from -1.5 to -11.6% (Shvartz, 1970; Moore, 1986); and DBP changes of from -8.5 to +18.6% (Ward et al, 1966; Moore, 1986).

Although the qualitative physiological response coincided with expected outcomes, quantitatively very little reproducibility of heart rate and blood pressure responses was seen either between subjects or in repeated tests of the same individual. Large variations in HR and BP values were seen in this sample of healthy young women. Other researchers have reported a similar variability in BP and HR results during application of orthostatic stress (Hyatt, Jacobson & Schneider, 1975; Moore & Newton, 1986). This disparity in results can be partially explained by differences in methodology. The experimental method used to induce orthostatic stress can alter the degree of hemodynamic response (Hyatt et al., 1975) but produce qualitatively similar changes in heart rate and blood pressure (Wolthuis et al., 1974). This study utilized a tilt table with foot support which circumvents the exercise reflex involved in active standing (Borst et al., 1982) but does involve minimal use of postural muscles.

The time interval at which the measurement is recorded must also be taken into account. In the

current study an effort was made to record immediate HR and BP response to acute orthostasis in the belief that major homeostatic adjustments to postural changes occur in the first minute (Spodick et al., 1971; Guyton, 1981). Measurements recorded later during upright posture might reflect adjustments to fluid loss from the microvasculature or increased venous pooling that occurs over time (Rowell, 1986).

Limitations of the Study

This study was limited by three principle factors. First, the small sample size probably did not allow for testing of a representative portion of the target population (Polit & Hungler, 1986, pg. 425). Second, the method used to simulate orthostasis may have allowed some confounding variables to emerge. Use of a tilt table with foot support was chosen in the belief that this method simulated more closely the experience a patient might have clinically. Even though the tilt table method avoided the exercise response involved in active standing, with the feet supported there was still minimal use of postural muscles. Muscular activity was noted in several subjects as they adjusted leg position to regain a feeling of balance after being tilted to an upright position. It has been demonstrated that moderate muscular activity will significantly alter the BP and HR response to orthostasis (Smith et al., 1987).

Additional evidence suggests that afferent signals from skeletal muscle fibers play a role in mediating a pressor response (Kniffki, Mense, & Schmidt, 1981). Smith et al.,(1987) similarly noted a positive correlation between electromyelographic activity and arterial blood pressure. The fact that 87% of the subjects in this study exhibited greatest HR and SBP changes in the second and third tilt suggest some form of anticipatory response and may corroborate findings that response to orthostasis is altered by contraction of abdominal or leg muscles (Borst et al, 1982).

A third limitation was the interval at which the first HR and BP measurements were recorded. Although measurement of HR was continuous, the first BP measurement was recorded at one minute into the tilt. There is evidence that the baroreceptor response occurs in the first 10 to 20 seconds of upright posture (Borst et al, 1982). It would be interesting to measure HR and BP much earlier and perhaps more frequently during the tilt procedure in an effort to capture the earliest "peak" of barorecptor response.

Summary and Implications for Nursing

The primary purpose of this study was to examine the relationship of physical fitness to HR and BP response to orthostasis. No fitness related differences could be demonstrated between three groups of healthy

women categorized on the basis of individual VO_2max. The only relationship that emerged was that of a strong inverse correlation between VO_2max and baseline heart rate; a relationship well documented in the literature.

The reflex homeostatic response to standing is particularly important to the hospitalized patient who is exposed to a variety of factors which could compound the risk of circulatory compromise. Orthostatic hypotension is a common problem among hospitalized patients (Memmer, 1988). The literature differentiates between two general categories of hypotension; that which is functional and usually reversible and that which is neurogenic in origin (Schatz, 1984). The list of circumstances which predispose the patient to functional orthostatic hypotension is lengthy. Factors commonly seen in the patient population such as advanced age, fever, fluid and electrolyte imbalances, pharmacological intervention, immobility, all put the patient at risk (Ziegler, 1980; Schatz, 1984).

The prolonged recumbency which is often characteristic in the treatment of critically ill patients is particularly detrimental to the maintenance of normal cardiovascular equilibrium (Winslow, 1985). Bed rest initiates a cascade of physiological mechanisms culminating in a depressor response (Blomqvist & Stone, 1983). A substantial reduction of blood volume along

with a reduction in peak oxygen uptake is followed by the need for a greater HR elevation to maintain BP during orthostasis (Blamick, Goldwater & Convertino, 1988).

Very little has been done relating physical fitness to the care given hospitalized patients. Goldwater et al. (1980) have suggested that athletic conditioning may predispose the hospitalized patient to orthostatic intolerance, because of lower circulating levels of catecholamines associated with chronic exercise. It should be noted that Goldwater et al. (1980) examined only males from 45 to 55 years old. Females were not included in this study. Therefore the results cannot be generalized to females or to a younger population. Schilling & Molen (1984), on the other hand, demonstrated a positive relationship between fitness level and measures of recovery including duration of hospital stay and patient self-evaluation of wellness in post-operative hysterectomy patients. It should be noted that physical fitness was measured by the patient's postexercise heart rate response to the Canadian Home Fitness Test and not in terms of VO_2max.

An understanding of factors impinging on the homeostatic reflex to postural change is imperative for health care personnel working in the critical care sector. Additional knowledge of the role physical

fitness may play in shaping and perhaps altering the human response to illness is needed.

Suggestions for Further Research

There is still a great deal to be learned about physical fitness and circulatory dynamics particularly as they apply to the hospitalized patient. The following are suggestions for research:

1) An examination of postural response in hospitalized patients before and after a prescribed course of exercise.

2) An examination of different levels and types of daily activity as they relate to recovery time measured by indices such as duration of hospital stay.

3) The relative importance of physical fitness levels prior to hospitalization for wound healing including cardiac ischemia.

4) The role of fitness and response to drug therapy.

5) A replication of this study with a different definition of fitness, for example, upper arm or abdominal strength.

BIBLIOGRAPHY

Astrand, P. O. & Rodahl, K. (1977). Textbook of work physiology; physiological basis of exercise (2nd ed.). New York: McGraw Hill.

Berne, R. M. & Levy, M. N. (1986). Cardiovascular Physiology. St. Louis: C. V. Mosby Company.

Blamick, C. A., Goldwater, D. J. & Convertino, V A. (1988). Leg vascular responsiveness during acute orthostasis following simulated weightlessness. Aviation, Space and Environmental Medicine, 59, 40-3.

Blomqvist, C. G., & Stone, H. L. (1983). Cardiovascular adjustment to gravitational stress. In Handbook of Physiology. The cardiovascular system, peripheral circulation and organ blood flow. (J. T. Sheperd, R. M. Abboud, & S. R. Geiger, eds.), sect. 2, vol. III, part 2, 1025-1063. Bethesda, Maryland: American Physiological Society.

Borg, G. V. & Lindholm, H. (1967). Perceived exertion pulse rate during graded exercise in various age groups. ACTA Med.Scand. (Suppl.), 474, 194.

Borst, W., Wieling, W., van Brederode, J. F. M., Honda, A., de Rijk, L. G. & Dunning A. J. (1982). Mechanisms of initial heart rate response to postural change. American Journal of Physiology, 243, H676-H681.

Brevetti, G., Chiariello, M. & Campanella, G. (1983). Different types of orthostatic hypotension. Acta Neurologica, 5(6), 441-446.

Bruce, R. A., Kusumi, R. & Hosmer, D. (1973). Maximal oxygen intake and nomographic assessment of functional aerobic impairment in cardiovascular disease. American Heart Journal, 85, 546-562.

Colby, A. O. (1977). What does smoking really do to the heart? Military Medicine, 46, 53-58.

American College of Sports Medicine (1986). Guidelines for exercise testing and perscription. Philadelphia: Lea & Febiger.

Convertino, V. A., Sather, T. M., Goldwater, D. J. & Alford, W. R. (1986). Aerobic fitness does not contribute to prediction of orthostatic intolerance. Medical Science in Sports and Exercise, 18(5), 551-556.

Convertino, V. A., Montgomery, M. S. & Greenleaf, M. S. (1984). Cardiovascular responses during orthostasis: effect of an increase in VO_2max. Aviation, Space, and Environmental Medicine, 55, 702-708.

Donald, D. E., & Shepherd, J. T. (1979). Cardiac receptors: normal and disturbed function. American Journal of Cardiology, 44, 873-878.

Epstein, M. (1978). Renal effects of head-out water immersion in man; implications for an understanding of volume homeostasis. Physiological Review, 58, 529-531.

Frank, M. J., & Alvarez-Mena, S. V. (1973). Cardiovascular physical diagnosis. Chicago: Year Book.

Frey, M. A. B. (1987). Considerations in prescribing preflight aerobic exercise for astronauts. Aviation, Space and Environmental Medicine, 58, 1014-1023.

Frey, M. A. B., Mathes, k. L. & Hoffler, G. W. (1987). Aerobic fitness in women and responses to lower body negative pressure. Aviation, Space and Environmental Medicine, 58, 1149-1152.

Gauer, O H., Henry, J. P. & Behn, C. (1970). The regulation of extracellular fluid volume. Annual Review of Physiology, 32, 547-595.

Gauer, O. H. & Thron, H. L. (1965). Postural changes in circulation. In: Handbook of Physiology, (W. F. Hamilton, ed), American Physiological Society: Washington, DC, sect. 2, 3, 2409-2439.

Glezer, G. A. & Moskalenko, N. P. (1972). The physiological and pathological haemodynamic changes in orthostasis in subjects with normal arterial pressure. Cor Vasa, 14(4), 265-277.

Goldwater, D. J., DeLada, M., Polese, A., Keil, L. & Luetcher, J. A. (1980). Effect of athletic conditioning on orthostatic tolerance after prolonged bedrest. Medical Science in Sports and Exercise, (abstract), III-287, 1100.

Guyton, A. (1981). Textbook of medical physiology. Philadelphia: W. B. Saunders.

Härmä, M. I. & Länsimies, E. (1985). Orthostatic tolerance at different times of the day in physically average and very fit dayworkers. Annals of Clinical Research, 17, 27-31.

Hill, D. W., Hill, J. S., Grisham, S. C. & Zauner, C. W. (1987). Plasma volume response to exercise on five consecutive days. Journal of Sports Medicine, 27, 6-10.

Hudson, D. L., Smith, M. L. & Raven P. B. (1987). Medicine & Science in Sports & Exercise 19, (4), 375-381.

Hyatt, K. H., Jacobson, L. B. & Schneider V. S. (1975). Comparison of 70° tilt, LBNP, and passive standing measures of orthostatic tolerance. Aviation, Space & Environmental Medicine, 46, (6), 801-808.

Ibrahim, M. M. (1975). Localization of lesion in patients with idiopathic orthostatic hypotension. British Heart Journal, 37, 868-872.

Ibrahim, M. M., Tarazi, R. C. & Dustan, H. P. (1975). Orthostatic hypertension: mechanisms and management. American Heart Journal, 90(4), 513-520.

Klein, K. E., Wegmann, H. M. & Kuklinski, R. (1977). Athletic endurance training--advantages for space flight?: the significance of physical fitness for selection and training of spacelab crews. Aviation, Space and Environmental Medicine, 48(3), 215-222.

Klein, K. E., Bruner, H., Jovy, D., Vogt, L. & Wegmann, H. M. (1969). Influence of stature and physical fitness on tilt-table and acceleration tolerance. Aerospace Medicine, 40(3), 293-297.

Klein, K. E., Wegmann, H. Bruner, H. & Vogt, L. (1969). Physical fitness and tolerances of environmental extremes. Aerospace Medicine, 40(9), 998-1001.

Kniffki, K. D., Mense, S. & Schmidt, R. F. (1981). Muscle receptors with five afferent fibers which may evoke circulatory reflexes. Circulation Research, 48(suppl. I), 25-31.

Lance, V. & Spodick. H. (1977). Postural effects on the noninvasive baselines of ventricular performance. Cardiology, 62, 296-304.

Marshall, R. J. & Shepard, J. T. (1968). Cardiac function in health and disease, Philadelphia: W.B. Saunders Co.

Matalon, S. V. & Farhi, L. E. (1979). Cardiopulmonary readjustments in passive tilt. Journal of Applied Physiology, 47(3), 503-507.

Memmer, M. K. (1988). Acute orthostatic hypotension. Heart & Lung, 17,(2), 134-140.

Polit, D. F. & Hungler, B. P. (1983). Nursing research; principles and methods. Philadelphia: J. B. Lippincott Company.

Raven P. B., Rohm-Young, D. & Blomqvist, G. C. (1984). Physical fitness and cardiovascular response to lower body negative pressure. Journal of Applied Physiology, 56(1), 138-144.

Rowell, L. B. (1986). Human circulation: regulation during physical stress, New York: Oxford University Press.

Rushmer, R. F. (1970). Cardiovascular Dynamics. Philadelphia: Saunders.

Schatz, I. J. (1984). Orthostatic hypotension: functional and neurogenic causes. Archives of Internal Medicine, 144, 773-777.

Schilling, J. A. & Molen M. T. (1984). Physical fitness and its relationship to postoperative recovery in abdominal hysterectomy patients. Heart & Lung, 13,(6), 639-644.

Shvartz, E. (1968). Relationship between endurance and orthostatic tolerance. Journal of Sports Medicine, 9, 75-80.

Shvartz, E. & Meyerstein, N. (1970). Tilt tolerance of young men and women. Aerospace Medicine, 41, 253-255.

Sjostrand, T. (1952). The regulation of the blood distribution in man. Acta Physiology Scandinavia, 26, 312-327.

Smith, M. L, Hudson, D. L. & Raven P. B. (1987). Effect of muscle tension on the cardiovascular responses to lower body negative pressure in man. Medicine and Science in Sports and Exercise, 19, (5), 436-442.

Smith, J. J., Hughes, C.V., Ptacin, M. J., Barney, J. A., Tristani, F. E. & Ebert, T. J. (1987). The Effect of age on hemodynamic response to graded postural stress in normal men. Journal of Gerontology, 42(4), 406-411.

Smith, M. L. & Raven, P. B. (1986). Cardiovascular responses to lower body negative pressure in endurance and static exercise-trained. Medicine and Science in Sports and Exercise, 18(5), 545-550.

Spodick, D. H., Meyer, & St. Pierre, J. R. (1971). Effect of upright tilt on the phases of the cardiac cycle in normal subjects. Cardiovascular Research, 5, 210-214.

Stegmann, J., Busert, A. & Brock, D. (1974). Influence of fitness on the blood pressure control system in man. Aerospace Medicine, 45(1), 45-48.

Sundkvist, G. & Lilja B. (1983). Effect of the degree and speed of tilt on the immediate heart rate reaction. Clinical Physiology, 3, 381-386.

Vander, A. J., Sherman, J. H. & Luciano, D. S. (1985). Human physiology; the mechanisms of body function (4th ed.) New York: McGraw-Hill.

Vargas, E. & Lye. M. (1983). Physiological responses to postural change in young and old healthy individuals. Experimental Gerontology, 17, 445-452.

Wang, Y., Marshall, R. J., & Sheperd, J. T. (1960). The effect of changes in posture and of graded exercise on stroke volume in man. Journal of Clinical Investigation, 39, 1051-1061.

Ward, R. J., Danziger, R., Bonica, J. J. Allen, G. D., & Tolas, A. G. (1966). Cardiovascular effects of change of posture. Aerospace Medicine, 37, 257-259.

Winder, W. W., Hagberg, J. M., Hickson, R. C., Ehsani, A. A. & McLane, J. A. (1978). Time course of sympathoadrenal adaptation to endurance exercise training in man. Journal of Applied Physiology, 45, (3), 370-374.

Winslow, E. H. (1985). Cardiovascular consequences of bed rest. Heart & Lung, 14, (3), 236-246.

Wolthuis, R. A., Bergman, S. A. & Nicogossian, A. E. (1974). Physiological effects of locally applied reduced pressure in man. Physiological Reviews, 54, (3), 566-593.

Woods, N. F. & Cantanzaro, M. (1988). Nursing research. St. Louis: C. V. Mosby Company.

Ziegler, M. G. (1980). Postural hypotension. Annual Review of Medicine, 31, 239-245.

Appendix A

Bruce Protocol Treadmill Test

Speed and gradient are raised every three minutes as follows:

Stage	Speed	Gradient	Duration	Mets
I	1.7 mph	10%	3 min	5
II	2.5 mph	12%	3 min	7
III	3.4 mph	14%	3 min	9-12
IV	4.2 mph	16%	3 min	12-14

Adapted from Bruce et al. (1973)

Appendix B
Screening Questionnaire

Please answer the following questions as accurately as possible. All information will remain anonymous and will be kept under lock and key by the reasearcher.

If you have any questions feel free to ask me for clarification or assistance. If at any time you should not want to answer you may refuse to do so.

---

1) What is your present age?.......................... .

2) Date of last menstrual period......................

3) What is your height?.............Weight?...................

4) Are you presently being treated for any health problems?
If yes please specify...
....
....

5) Have you ever been treated for any poblems involving:

yes no
- a) lungs..............................
- b) heart..............................
- c) gastrointestinal (digestive) system......
- d) vascular system (veins)................
- e) endocrine (hormone) system..............
- f) elimination...........................
- g) bones or joints........................
- h) balance...............................

If the answer to any of the above was yes. please explain:..
....

6) Have you ever been treated for any psychological or emotional problems?
....

7) Have you ever been told that you have high blood pressure?...... Low blood pressure?..........

8) Have you ever fainted?......
If yes, what were the circumstances................
....

9) Are you presently a smoker?.............

10) How would you rate your current state of physical fitness?

Very poor Poor Fair Good Very good Excellent

11) How frequently do you participate in a physical activity (sport or exercise) in which you work up a sweat for a period of at least 30 minutes? (check one of the following):

.....a) At least once daily
.....b) Every other day
.....c) Twice a week
.....d) Once a week
.....e) Once every two weeks
.....f) Once a month
.....g) Less than once a month

12) Do you participate in a routine form of exercise?..........
If yes, please describe...........................

....

....

Appendix C

DESIRABLE WEIGHTS FOR WOMEN

According to Height and Frame, Ages 25 and Over

Weight In Pounds Including Indoor Clothing*

Height (with 2" heals)	Small Frame	Medium Frame	Large Frame
4'10"	92-98	96-107	104-119
11"	94-101	98-110	106-122
5' 0"	96-104	101-113	109-125
1"	99-107	104-116	112-128
2"	102-110	107-119	115-131
3"	105-113	110-122	118-134
4"	108-116	113-126	121-138
5"	111-119	116-130	125-142
6"	114-123	120-135	129-146
7"	118-127	124-139	133-150
8"	122-131	128-143	137-154
9"	126-135	132-147	141-158
10"	130-140	136-151	145-163
11"	134-144	140-155	149-168
6" 0"	138-148	144-159	153-173

*Indoor clothing = 3lbs

Subtract 1lb for each year under 25

From the Metropolitan Life Insurance Company

Statistical Bulletin, October, 1977.

Appendix D
College of Sports Medicine
Guidelines for Exercise Testing

	APPARENTLY HEALTHY		*HIGHER RISK*			*WITH DISEASE*
	Below 45	*45 and Above*	*Below 35 No Symptoms*	*35 and Above No Symptoms*	*Symptoms*	*Any Age*
Maximal Exercise Test Recommended Prior to an Exercise Program	No	Yes	No	Yes	Yes	Yes
Physician Attendance Recommended for Maximal Testing	No (under 35)	Yes	Yes	Yes	Yes	Yes
Physician Attendance Recommended for Sub-maximal Testing	No	No	No	Yes	Yes	Yes

Appendix E

CONSENT FORM

UNIVERSITY OF WASHINGTON

CONSENT FORM

The Effect of Physical Fitness on Response to Orthostasis

INVESTIGATORS:

CAROLYN K. GOOCH, R.N., B.S N.
Graduate Student
Physiological Nursing
365-0465

William Ryan, Ph.D., R.N.
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Physiological Nursing
543-8650

PURPOSE AND BENEFITS

You are being asked to participate in a study to evaluate your heart rate and blood pressure response to moving from a lying down position to an upright posture. The purpose of the study is to examine the relationship between physical fitness and the cardiovascular response to standing.

You will directly benefit by receiving a free scientific measurement of your aerobic fitness level. Indirectly, you will be contributing to the understanding of physical fitness as it relates to cardiovascular stability.

PROCEDURES

The investigator will ask you several health related questions over the phone to determine whether you are eligible to participate. After eligibility is determined you will be sheduled for a test to measure your aerobic fitness level and given verbal instructions regarding preparation for the procedure. This test involves walking on a exercise treadmill at increasing levels of effort until you reach a point of excessive shortness of breath or are unable to continue. Heart rate will be measured by electrocardiogram and blood pressure by the standard cuff method throughout the test. You will breath into a machine that collects expired air. The procedure will be conducted at the pulmonary functions lab at University Hospital and will take from 45 minutes to one hour.

The second phase of the study involves a test of your heart rate and blood pressure response to upright posture. The procedure will take approximately one hour and will be conducted on a day following the exercise test at the School of Nursing in the Health Care Sciences Building on the University of Washington campus. You will

receive written instructions on how you should prepare prior to the procedure. You will be asked to lie on your back on a padded tilt table. A blood pressure cuff will be placed on one arm and electrocardiogram (EKG) leads (small sticky pads) will be placed on your chest in three different spots. These connections do not pierce the skin or cause discomfort in any way.

After connection of the measurement devices the table will be tilted until you are in a standing position. The table is equipped with a foot support and restraining straps so that you will remain safely and comfortably on the table. After you have been in the upright position for five minutes you will be returned to a horizontal position and the experimental phase is over. The investigator will record heart rate and blood pressure at several intervals before, during and after the upright tilting maneuver.

RISKS, STRESS AND DISCOMFORT

Although risks during exercise test are minimal there exists a possibility of abnormal blood pressure response, fainting, disorders of the heart beat (too fast, too slow or ineffective), and very rare instances of cardiac arrest. Every effort will be made to minimize these by preliminary testing and by observations during testing. Because the test consists of maximum physical exertion over a short period of time (9 to 12 minutes) you will be very fatigued and breathless when it is finished and may experience some residual muscle soreness. Emergency equipment and trained personnel will be available to deal with unusual situations should they arise.

No adverse effects are expected during the tilting maneuver. There is a possibility of inadequate cardiovascular response resulting in dizziness or fainting. In the event of abnormal responses of heart rate or blood pressure, the tilt will be immediately terminated. The investigator will deal with minor adverse reactions on the spot. Emergency personnel will be called in the unlikely event of severe cardiovascular problems.

OTHER INFORMATION

At no time during the study will photographs or audiotapes be used. All data collected will be kept strictly confidential under lock and key by the primary investigator. No individuals will have access to the information apart from the primary investigator, a statistician and thesis committee. This study is being done in partial fulfillment of the graduate requirements of the Department of Physiological Nursing. Study data in

the form of a thesis will be placed in the Univirsity of Washington library. Data gathered will be kept indefinitely. You are free to decide whether or not to participate in the study and are free to withdraw from the study at any time without penalty or loss of benefits to which you are otherwise entitled.

In the event of physical injury as a direct result of study procedures, subjects will be referred for appropriate treatment. Costs for treatment will be covered within the limits of the University of Washington compensation plan.

Signature of investigator Date

Subject's Statement

The study described above has been explained to me. I voluntarily consent to participate in this activity. I have had an opportunity to ask questions. I understand that future questions I may have about the research or about my rights as a subject will be answered by the investigator listed above.

Signature of subject Date

Appendix F
Subject Instructions

The following are guidelines for you to follow prior to and on the day of the procedure (exercise test/tilting maneuver) for which you are scheduled:

1. Do not take any medication or other drugs (except for birth control pills) for 72 hours prior to the test.

2. Obstain from drinking alcoholic beverages for 72 hours prior to testing.

3. Do not drink or eat for four hours prior to testing.

4. Do not participate in vigorous exercise for at least 24 hours prior to testing.

5. Report to the (Health Care Sciences building/pulmonary function lab) dressed in comfortable exercise clothes with tennis shoes or running shoes on.

Additional preparation for the tilt maneuver only:

1. No table salt or salty foods 24 hours prior to tilting.

2. No more than 2 eight ounce cans of carbonated drinks 24 hours prior to testing.

3. Keep a diary of all ingested foods and liquids for the day prior to testing and bring it with you on the day of the tilt maneuver.

If your have any questions regarding these instructions or any other aspect of the study please feel free to call the investigator at any time. You will be contacted by the investigator with directions to the testing areas.

INVESTIGATOR:

Carolyn K. Gooch, Graduate Student, School of Nursing
Home phone: 365-0465

Appendix G
Data Gathering Tool

Subject ________________
Date ____________
Baseline BP ____________
Baseline HR ____________

HEAD UP TILT

Heart Rate

1 min ____________

3 min ____________

5 min ____________

Blood Pressure

1 min ______________

3 min ______________

5 min ______________

Subjective complaints:

Adverse reactions:

Supine Rest Period

Heart Rate

5 min _____________

10 min _____________

Blood Pressure

5 min ______________

10 min ______________

Appendix H

Analysis of Variance

for Baroreceptor Sensitivity Index Between Groups

Source	D.F.	Sum of Squares	Mean Squares	F Ratio	F Probability
Between Groups	2	.3236	.1618	.0338	.9668
Within Groups	13	62.1928	4.7841		
Total	15	62.5164			

Appendix I

T-test of Baroreceptor Response Index

Between High-Fit and Low-Fit Groups

	Number of Cases	Mean	Standard Deviation	Standard Error
Group 1	4	1.2475	.630	.315
Group 2	4	1.0525	.439	.220

		Pooled Variance Estimate			Separate Variance Estimate		
F Value	2-Tail Prob.	t Value	Degree of Freedom	2-Tail Prob.	t Value	Degrees of Freedom	2-Tail Prob.
2.06	.569	.51	6	.630	.51	5.36	.632

Appendix J

Kruskal-Wallis Oneway ANOVA of Baroreceptor Response Index Between High-Fit, Med-Fit and Low-Fit Groups

Mean Rank	Cases	
9.75	4	GROUP = 1
8.13	8	GROUP = 2
8.00	4	GROUP = 3
	16 Total	

			Corrected for Ties	
CASES	Chi-Square	Significance	Chi-Square	Significance
16	.3695	.8313	.3695	.8313

Appendix K

Kruskal-Wallis Oneway ANOVA

of Baroreceptor Response Index

Between High-Fit and Low-Fit Groups

Mean Rank	Cases	
5.00	4	GROUP = 1
4.00	4	GROUP = 3
	8 Total	

			Corrected for Ties	
CASES	Chi-Square	Significance	Chi-Square	Significance
8	.3333	.5637	.3333	.5637